




# **Armed Forces College of Medicine AFCM**



# **Cerebrovascular Diseases**

**By**

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Let's make this day  
beautiful.

*Good Morning!*

W. W.

# INTENDED LEARNING OBJECTIVES (ILO)



**By the end of this lesson the student will be able to:**

- 1. Discuss cerebrovascular diseases.**
- 2. Describe pathological changes of cerebral infarction.**
- 3. Identify types of intracranial haemorrhage.**
4. Recognize hypertensive cerebrovascular diseases.
5. Determine types of Cerebral Aneurysms.
6. Analyse given data to diagnose pathological conditions of cerebrovascular diseases based on given clinical, radiologic data and/or laboratory findings

# Cerebrovascular Diseases



The term cerebrovascular disease denotes any abnormality of the brain caused by a pathologic process involving blood vessels.

**The three basic processes are**

- (1)Thrombotic occlusion of vessels
- (2) Embolic occlusion of vessels
- (3) Vascular rupture.

# Cerebrovascular Diseases



- **Thrombosis and embolism** cause ischemic injury or infarction of specific regions of the brain, depending on the vessel involved.
- **Hemorrhage** accompanies rupture of vessels, leading to direct tissue damage as well as secondary ischemic injury.
- **"Stroke"** is the clinical designation that applies to all these conditions, particularly when symptoms begin acutely.

# Stroke



- Stroke is a medical condition in which **poor blood flow** to the brain causes **cell death**.
- **There are two main types of stroke:** **ischemic**, due to lack of blood flow and **hemorrhagic** due to bleeding.
- **The clinical features:** of stroke result from focal cerebral ischemia.
- **Risk factors:** are those for **atherosclerosis, and hypertension**.

# Cerebral Infarction



- It results from cerebral arterial occlusion.
- The cause may be a **thrombus** or an **embolus**.

## ❖ Infarcts can be divided into two broad groups:

- 1- **Hemorrhagic infarcts** when there is reperfusion of ischemic tissue, either through collaterals or after dissolution of emboli.
- 2- **White infarcts** result from acute vascular occlusions.



# Cerebral Infarction



## Thrombotic infarction:

- It is due to thrombotic occlusion in the setting of **atherosclerosis of the cerebral arteries.**
- Thrombotic infarction is characteristically an **anemic (white) infarct.**

# Cerebral Infarction



**Embolic infarction:** more common and produces a **hemorrhagic infarction**

- **Source of emboli:**
  - **Cardiac mural thrombi** as in myocardial infarction.
  - **Thromboemboli from arteries**, most often from atheromatous plaques within internal carotid or arch of aorta.
  - **Paradoxical embolism** from deep leg veins which cross to arterial circulation through cardiac defects.
- **The middle cerebral artery** is the most frequent site because it is a direct extension of the internal carotid

# Gross and Microscopic Changes Associated with Cerebral Infarction



Time	Gross Changes	Microscopic Changes
0–12 h	No changes	Minimal or no changes
12–24 h	Minimal changes	Red (hypereosinophilic) neurons with pyknotic nuclei
24–48 h	Indistinct gray-white matter junction	Neutrophilic infiltration
2–10 d	Friable tissue with marked edema	Histiocytic infiltration; neurons disappear
2–3 wk	Tissue liquefies	Liquefactive necrosis; histiocytes filled with products of myelin breakdown
3 wk–mo	Fluid-filled cavity demarcated by gliotic scar	Fluid-filled cavity; reactive astrocytes and lipid-laden macrophages
Years	Old cyst surrounded by gliotic scar	Astrogliosis surrounding a cyst

# Cerebral Infarction



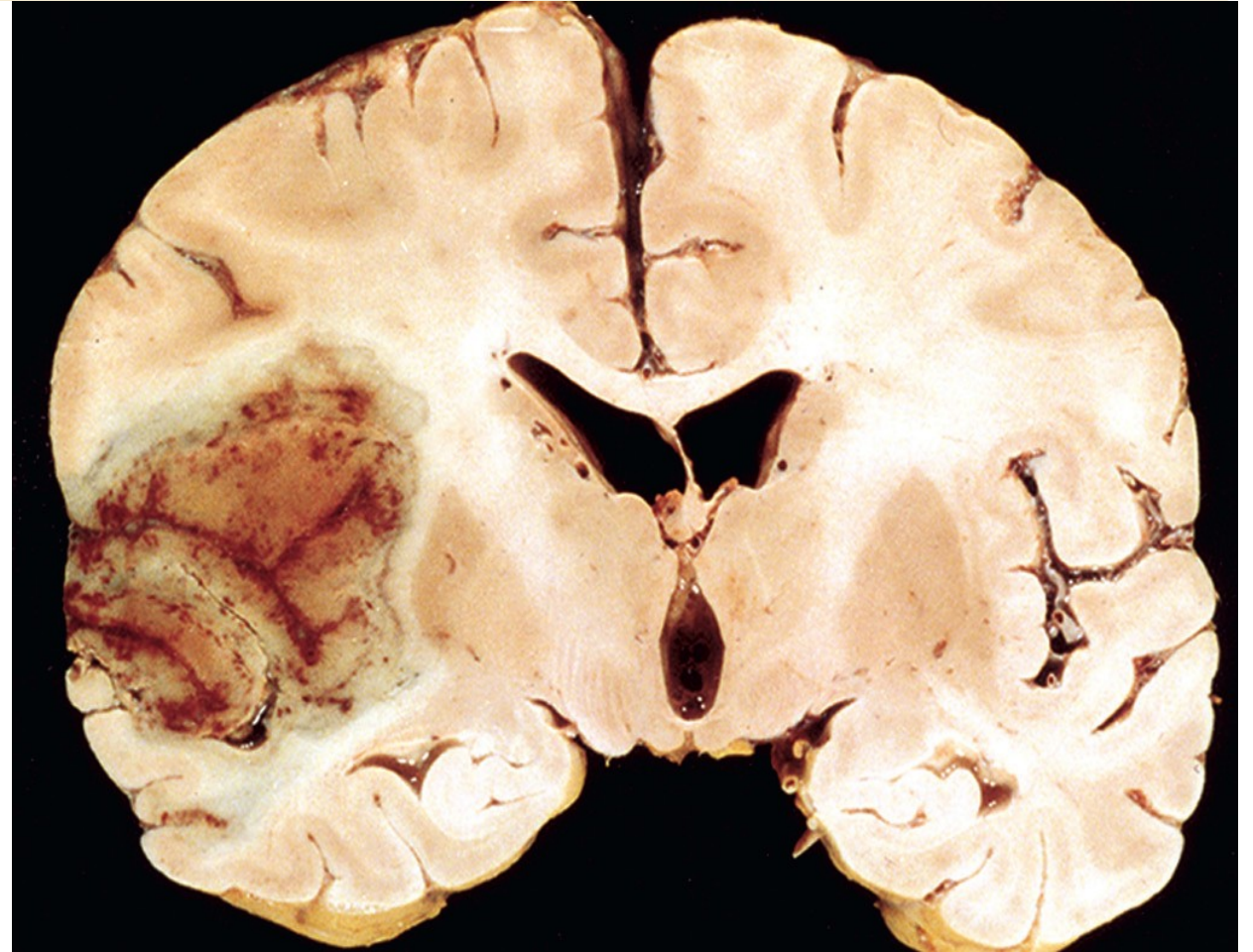
## Hemorrhagic infarcts:

- Usually manifest as multiple, petechial hemorrhages.
- **The microscopic picture** of hemorrhagic infarction is like those of ischemic infarction, **with the addition of blood extravasation.**

# Cerebral Infarction



Section of the brain showing a large, discolored, **focally hemorrhagic region in the left middle cerebral artery distribution (hemorrhagic, or red, infarction).**



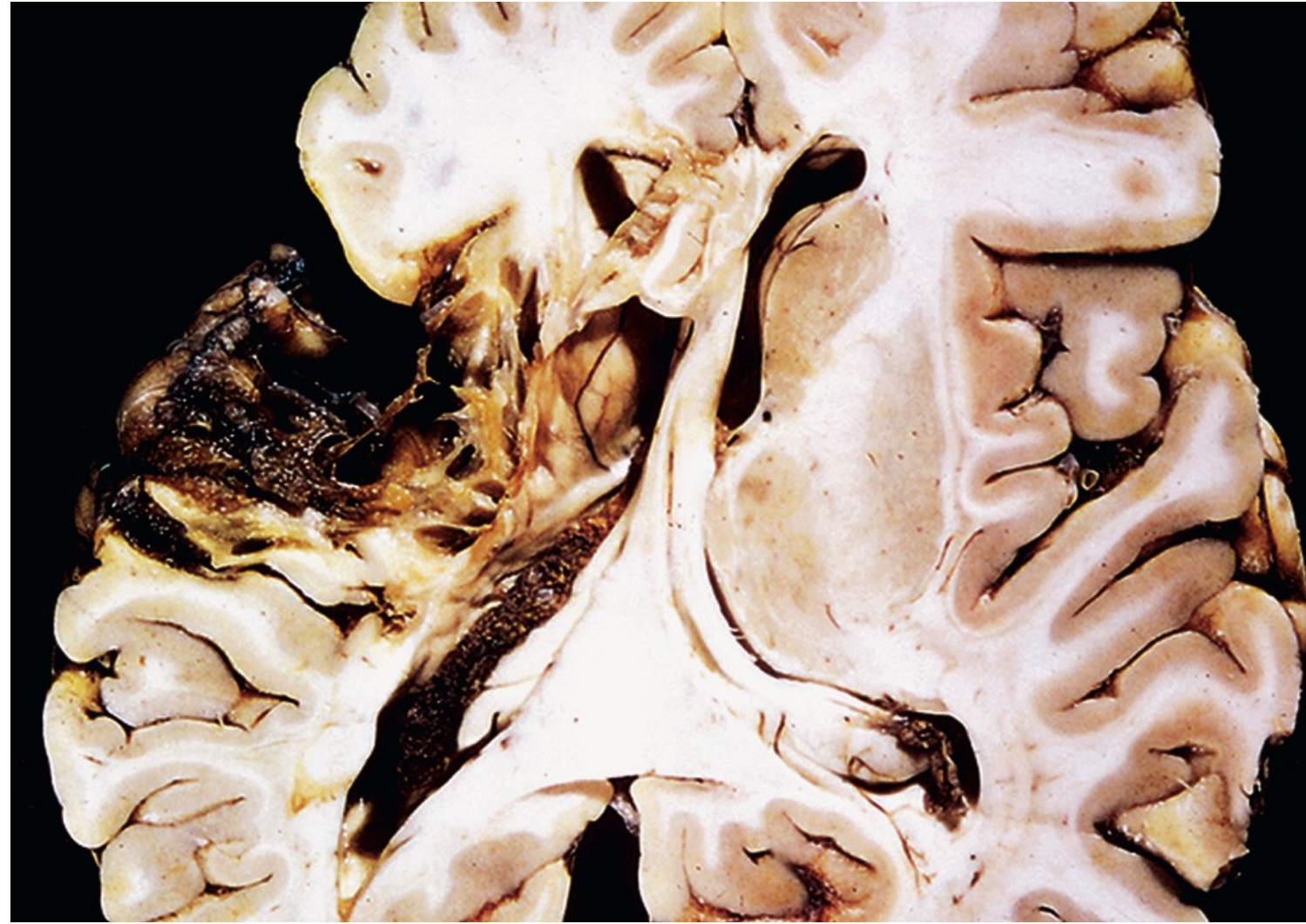
Robbins basic pathology, 10<sup>th</sup> edition, 2018



# Cerebral Infarction



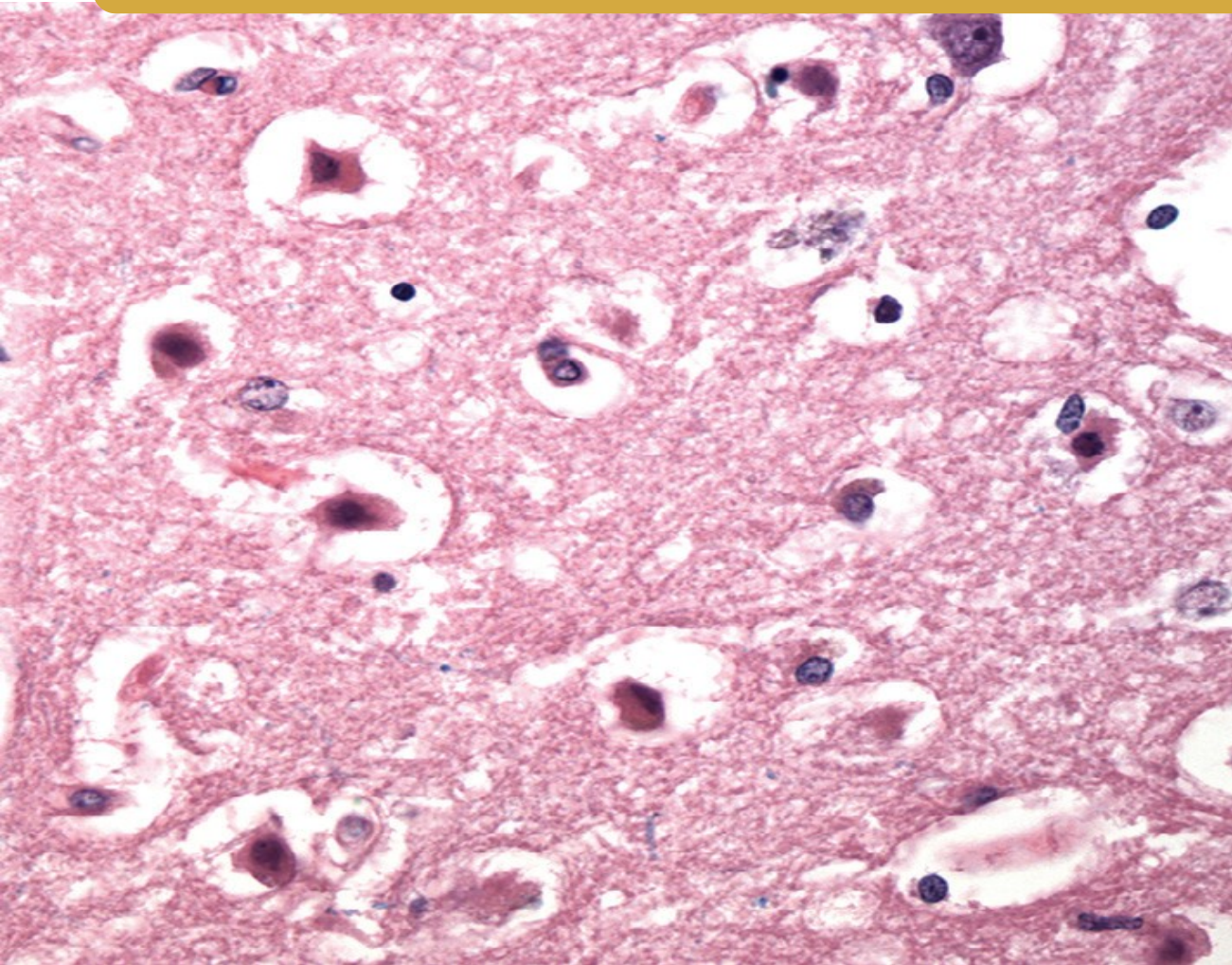
**Old cystic infarct:**  
shows destruction of  
cortex and surrounding  
gliosis.



Robbins basic pathology, 10<sup>th</sup> edition,  
2018

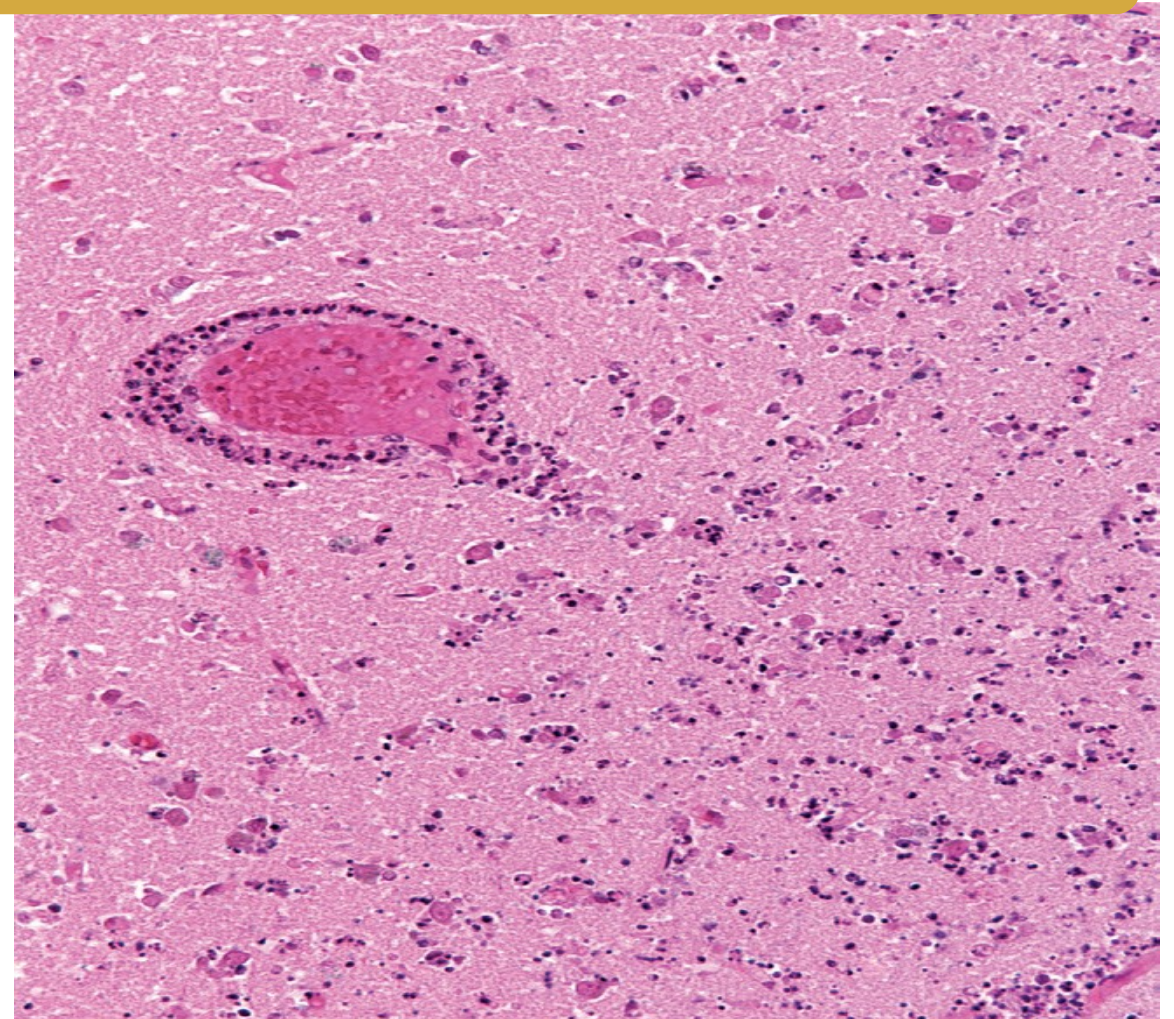


# Cerebral Infarction



Acute hypoxic-ischemic injury in the cerebral cortex. The cell bodies are shrunken and eosinophilic ("red neurons"), and the nuclei are pyknotic.

Robbins basic pathology, 10<sup>th</sup> edition, 2018



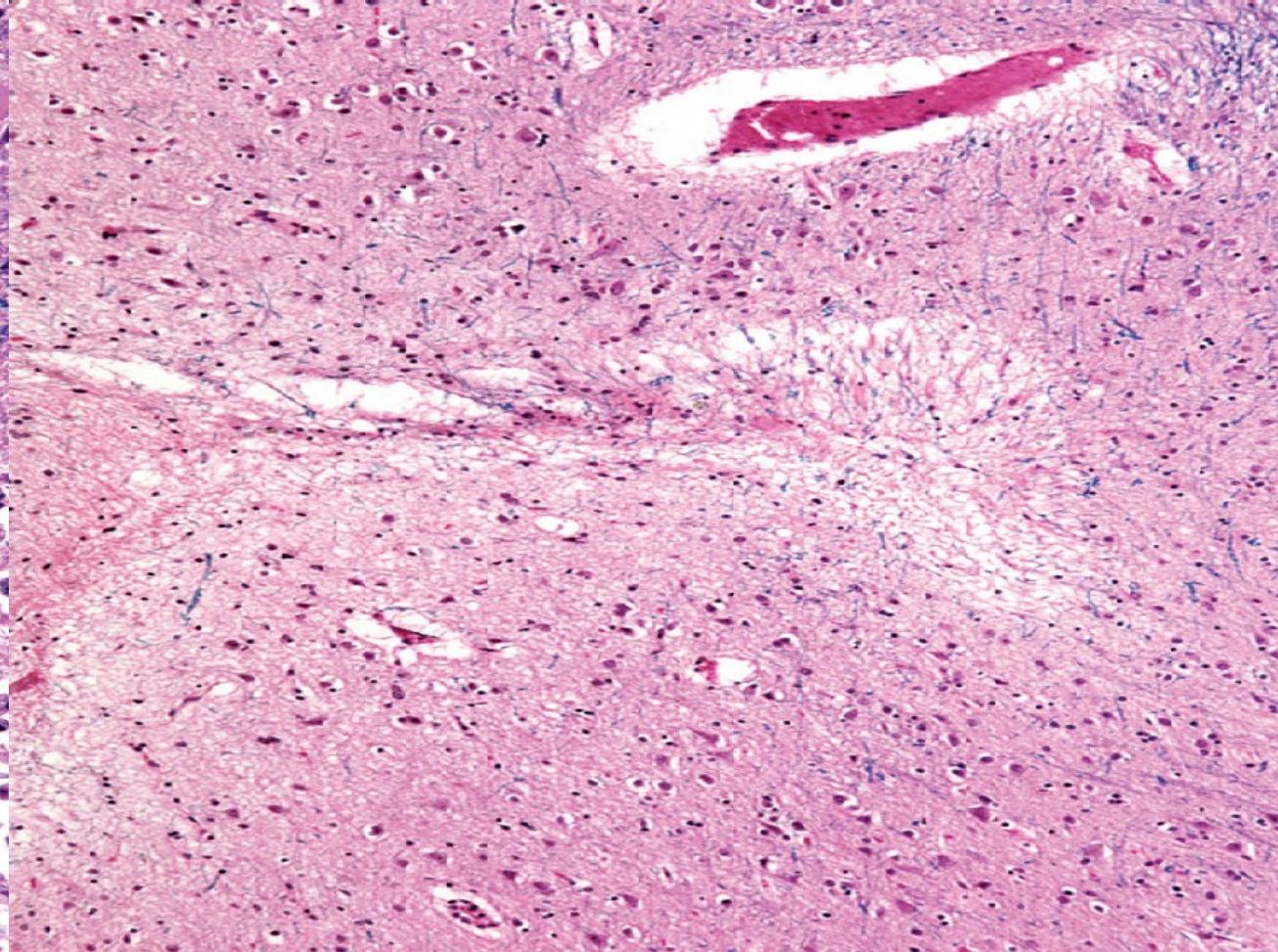
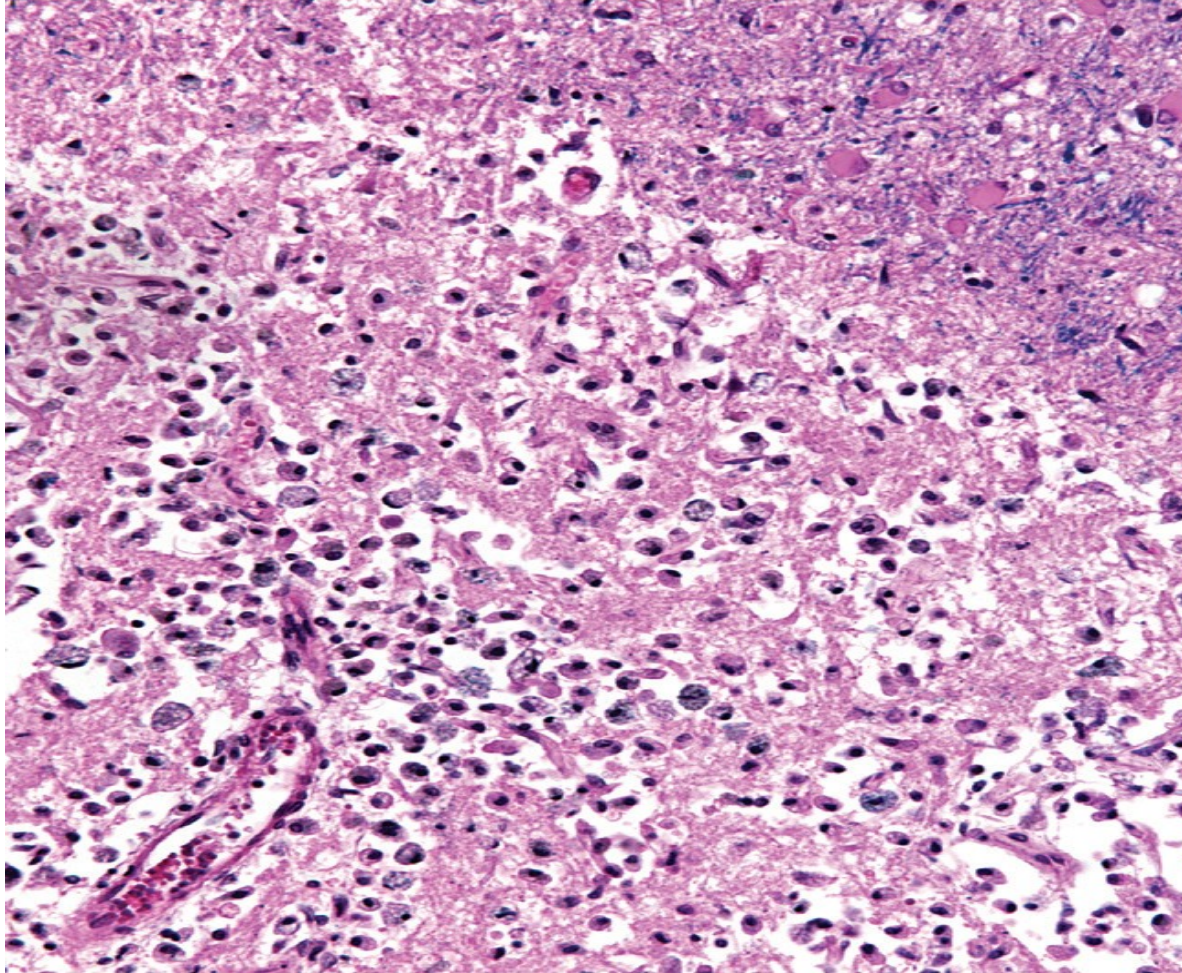
Infiltration of a cerebral infarction by neutrophils begins at the edges of the lesion, where the vascular supply is intact.

Neuroscience Module

Robbins basic pathology, 10<sup>th</sup> edition, 2018



# Cerebral Infarction



By day 10, an area of infarction shows the presence of macrophages and surrounding reactive gliosis.

Old intracortical infarcts are seen as areas of tissue loss and residual gliosis.

Robbins basic pathology, 10<sup>th</sup> edition,  
2018

Robbins basic pathology, 10<sup>th</sup> edition,

Neuroscience Module



# Intracranial Haemorrhage



- Haemorrhage into the brain may be **traumatic, nontraumatic, or spontaneous.**

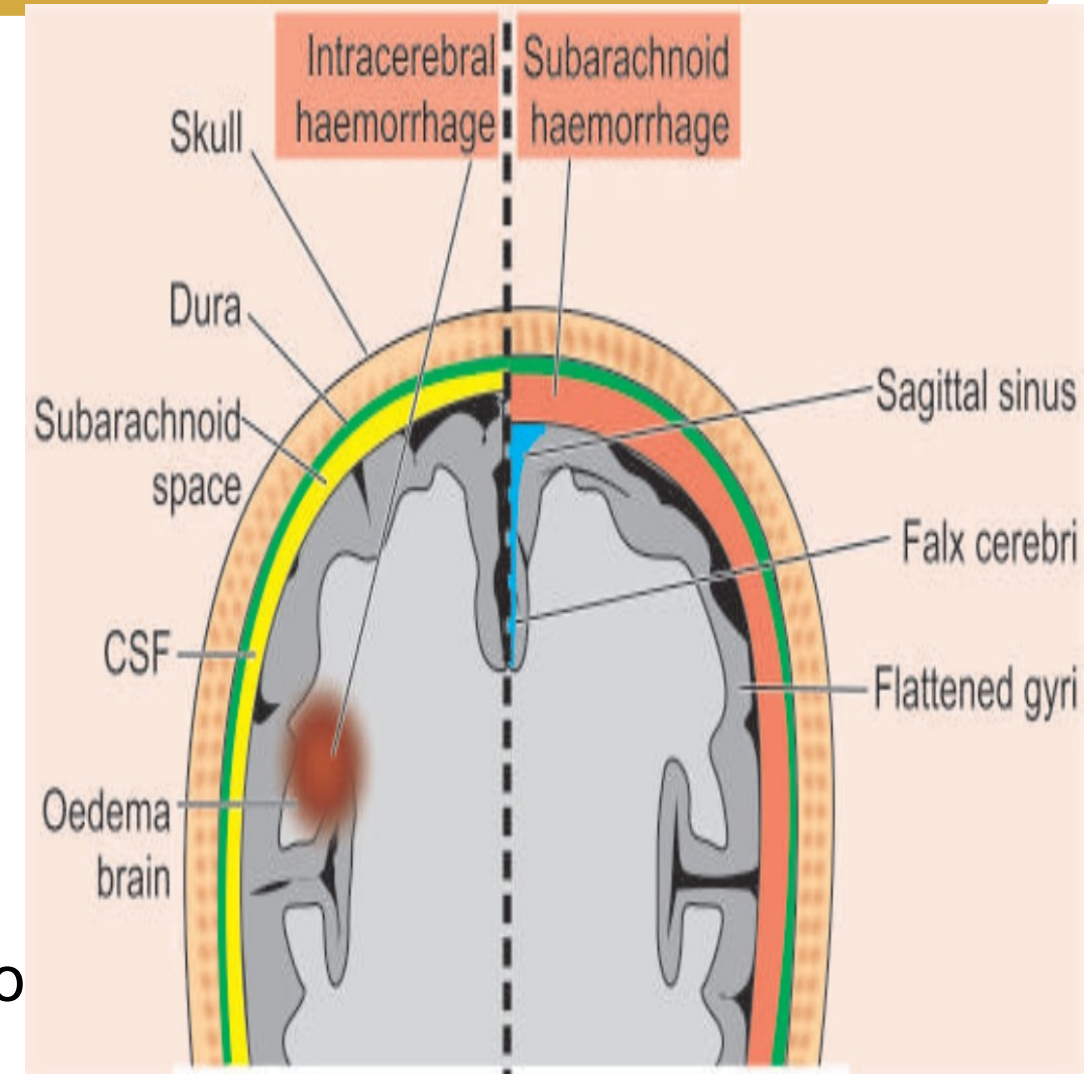
- **There are two main types of spontaneous non-traumatic intracranial haemorrhage:**

## 1. Intracerebral haemorrhage:

(usually of **hypertensive origin**)

## 2. Subarachnoid haemorrhage:

(commonly **aneurysmal in origin**), but also occur with other vascular malformations.



Harsh mohan, 7<sup>th</sup> edition, 2015

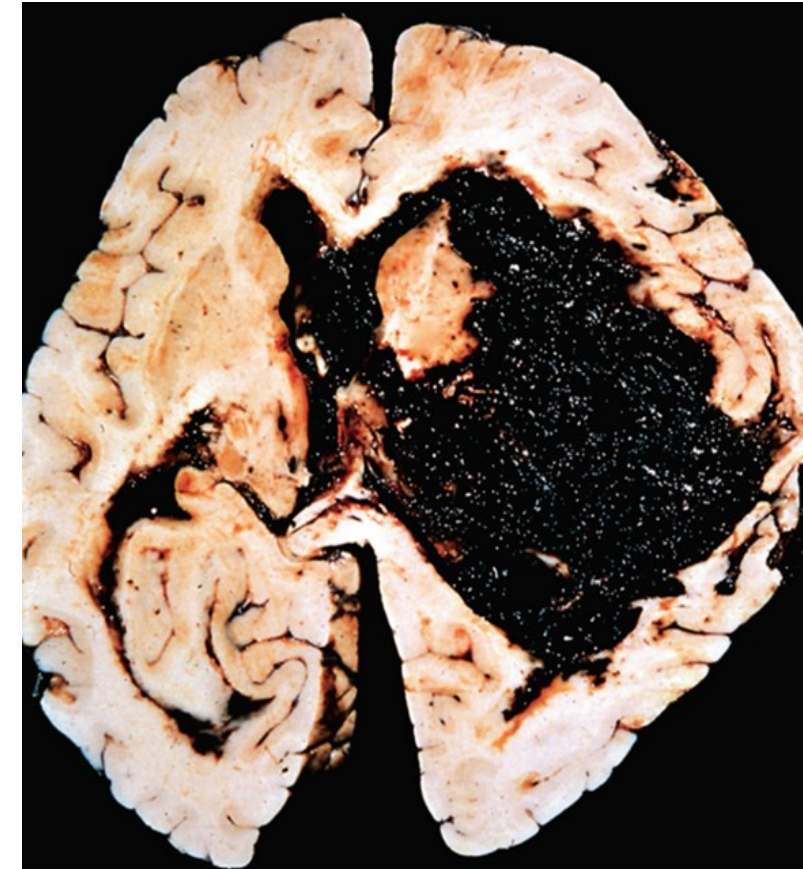
# Intracerebral Haemorrhage



- Spontaneous intracerebral hemorrhage has **a peak incidence at age of 60.**
- **Hypertension** is the leading cause.
- Typically occur in the basal ganglia (80%), thalamus, pons and cerebellum and cerebral cortex.

## Morphology:

- Acute hemorrhages consist of **extravasated blood (hematoma) compressing the parenchyma.**
- If patient survives, absorption of hematoma occurs **leaving a fluid-filled cystic cavity with a gliotic wall.**



Robbins basic pathology, 10<sup>th</sup> edition, 2018

# Intracerebral Haemorrhage



## Microscopic examination:

- **Early lesions:** consist of clotted blood surrounded by edematous brain tissue containing **neurons and glia displaying morphologic changes typical of anoxic injury.**
- **Later :** the edema resolves, **pigment- and lipid-laden macrophages appear, and proliferation of reactive astrocytes** becomes visible at the periphery of the lesion.

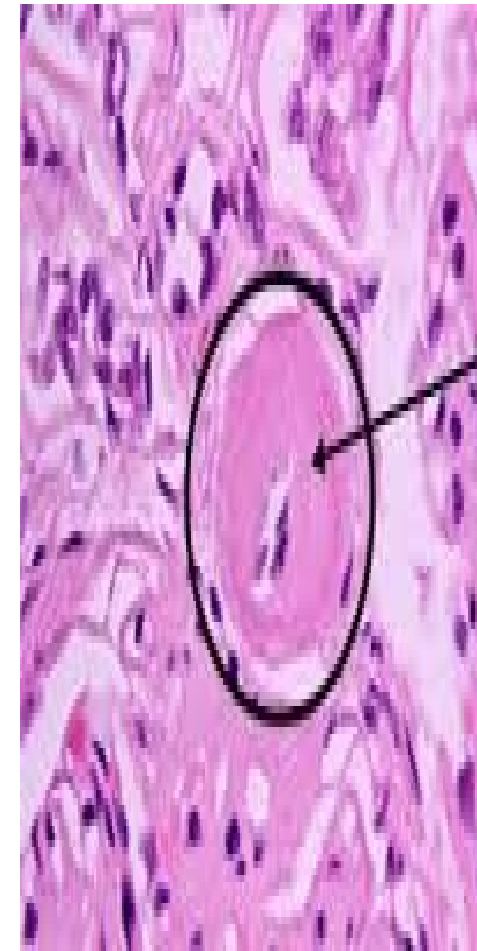
# Subarachnoid Haemorrhage

- Due to rupture of a **saccular (berry) aneurysm** on the circle of Willis.
- **Other causes** include rupture of vascular malformations and trauma
- **Clinical picture:** sudden onset of severe headache, often described as the "worst headache of my life.
- **Hydrocephalus** can occur in the acute stage or later in survivors due to fibrosis and obliteration of the subarachnoid space.

# Hypertensive cerebrovascular disease



- **Hypertension** causes **hyaline arteriolar sclerosis** of the deep penetrating arteries and arterioles that supply the basal ganglia, the hemispheric white matter, and the brain stem.
- Affected arteriolar walls are **weakened and are more vulnerable to rupture.**



The thick pink ring surrounding the light pink lumen represents the hyaline deposits.

[anatomy.elpaso.ttuhsu.edu](http://anatomy.elpaso.ttuhsu.edu)

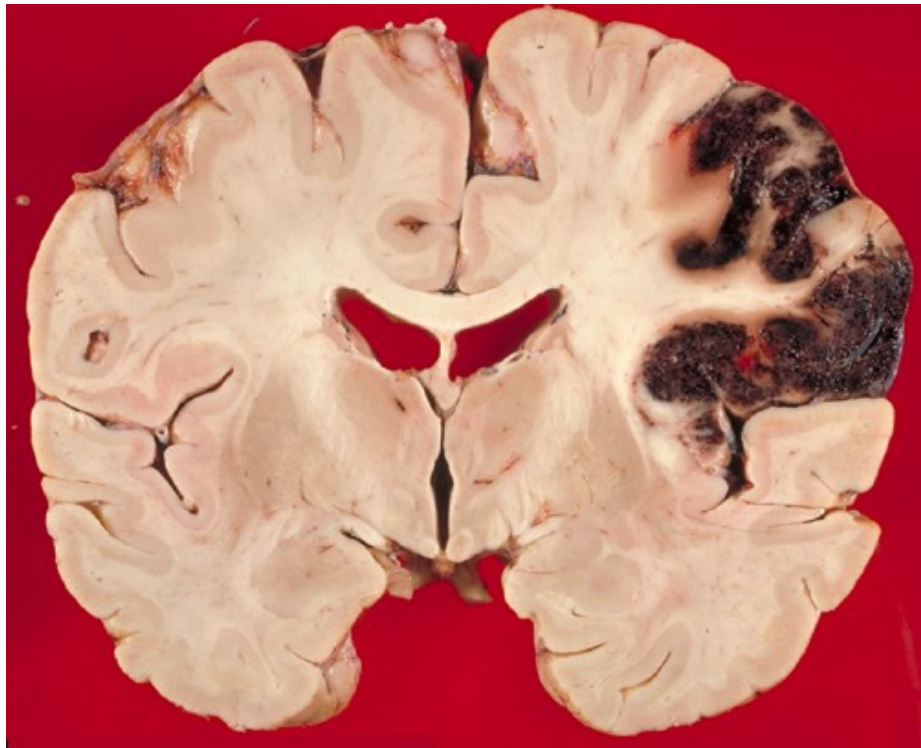


# Pathologic processes related to Hypertension



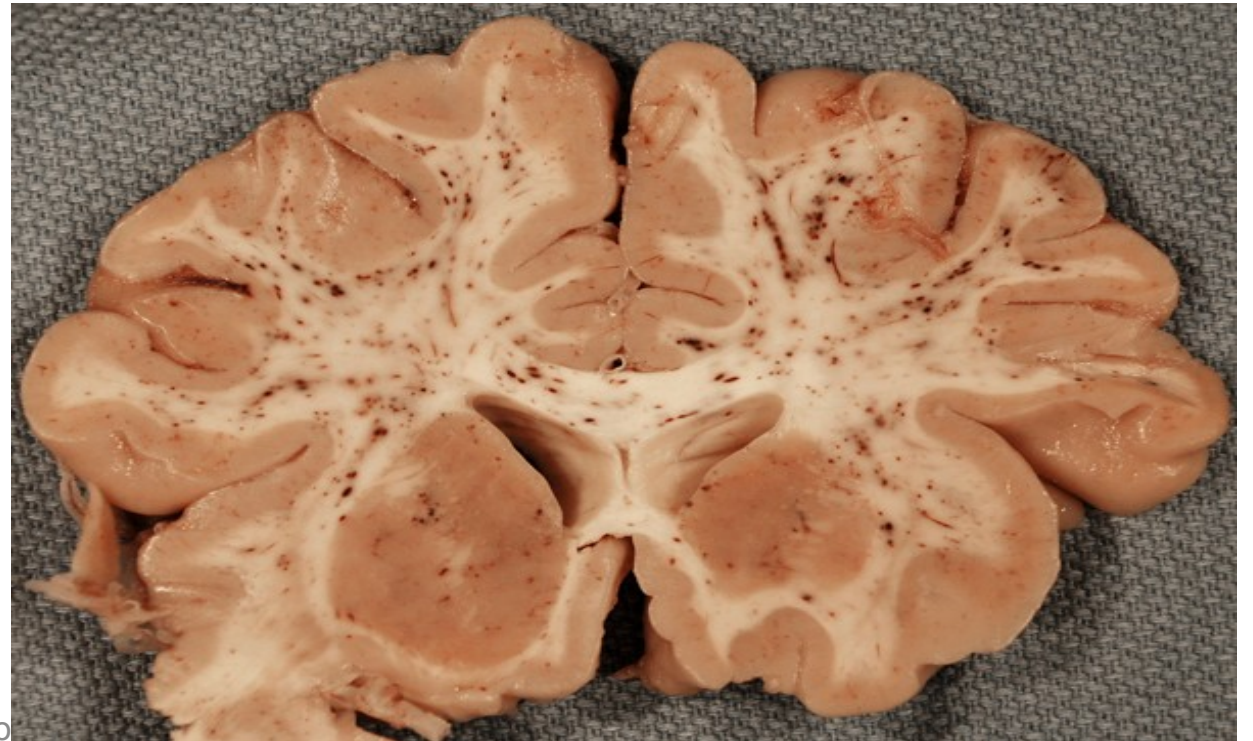
## ❖ Intracerebral hemorrhage:

Acute massive hemorrhage & stroke.



## ❖ Lacunar infarcts:

**Small, cavitory** infarcts of the deep grey matter in the basal ganglia and thalamus due to occlusion of **single**

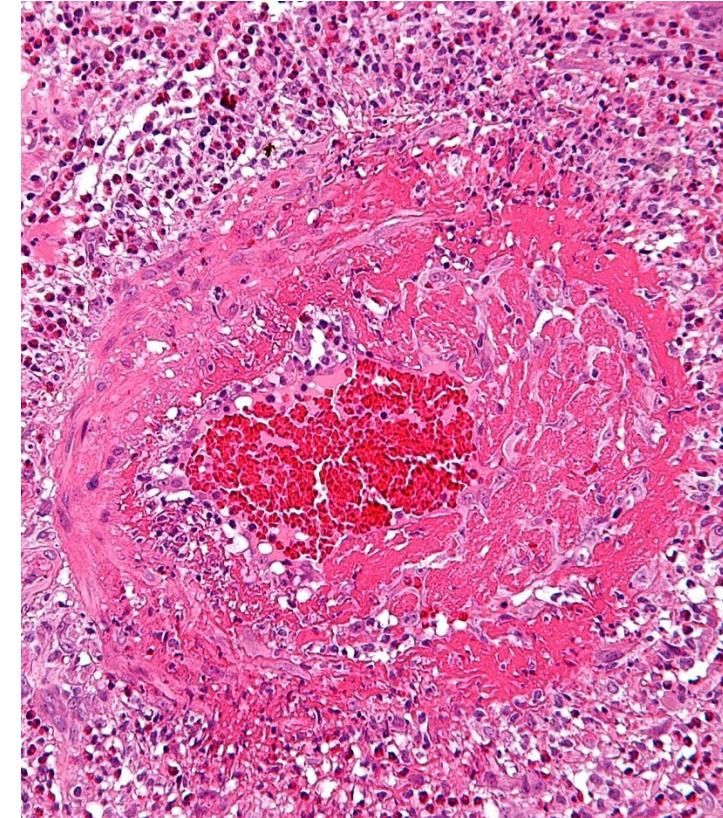


Neuro

# Hypertensive Encephalopathy



- It is a condition characterized by **high blood pressure and neurological symptoms** including **headache, confusion, vomiting, convulsions up to coma.**
- The sudden **sustained rise of blood pressure overwhelms autoregulatory mechanisms of brain lead to permeability of blood brain barrier and brain edema**
- **Post mortem picture shows:**
- ❖ **Cerebral edema grossly and fibrinoid necrosis of arterioles may be seen microscopically.**



[https://upload.wikimedia.org/wikipedia/commons/thumb/3/34/Churg-Strauss\\_syndrome\\_-\\_high\\_mag.jpg/1920px-Churg-Strauss\\_syndrome\\_-\\_high\\_mag.jpg](https://upload.wikimedia.org/wikipedia/commons/thumb/3/34/Churg-Strauss_syndrome_-_high_mag.jpg/1920px-Churg-Strauss_syndrome_-_high_mag.jpg)

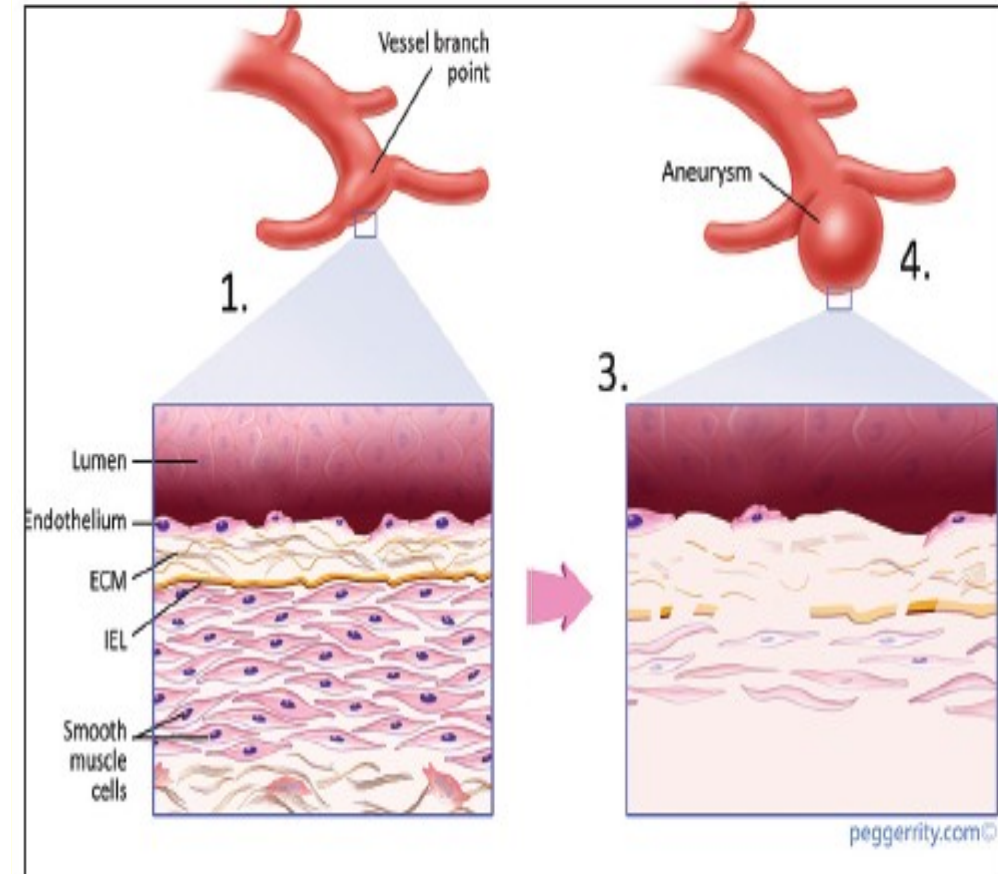


# Cerebral aneurysms



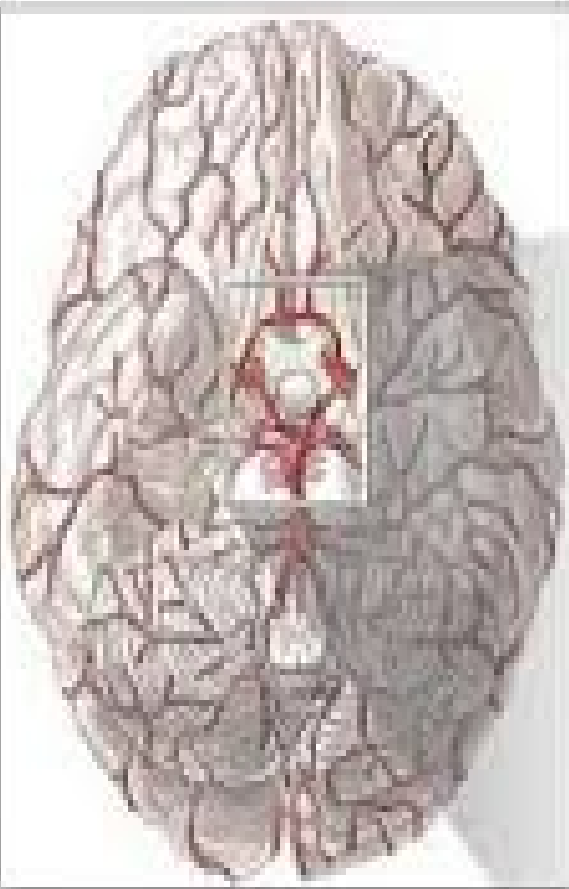
## 1- Congenital Berry aneurysms

- Multiple small aneurysms
- Due to congenital absence of the media
- At the bifurcation of cerebral arteries **in the circle of Willis.**
- Rupture is precipitated by a **sudden increase in blood**



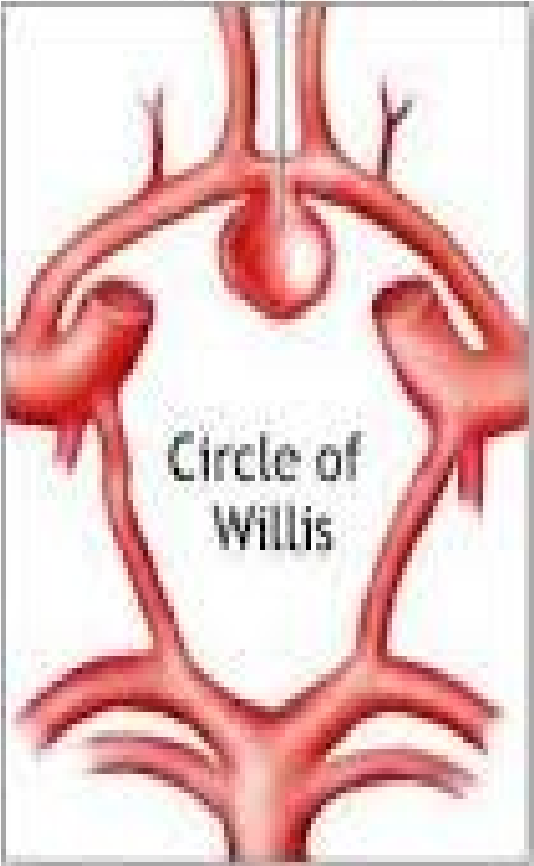
<https://oaepublishstorage.blob.core.windows.net/8e33f7b3-b022-4f77-9c56-14ee4d21aba4/226.fig.1.png>





Bottom view of brain and major arteries of the brain

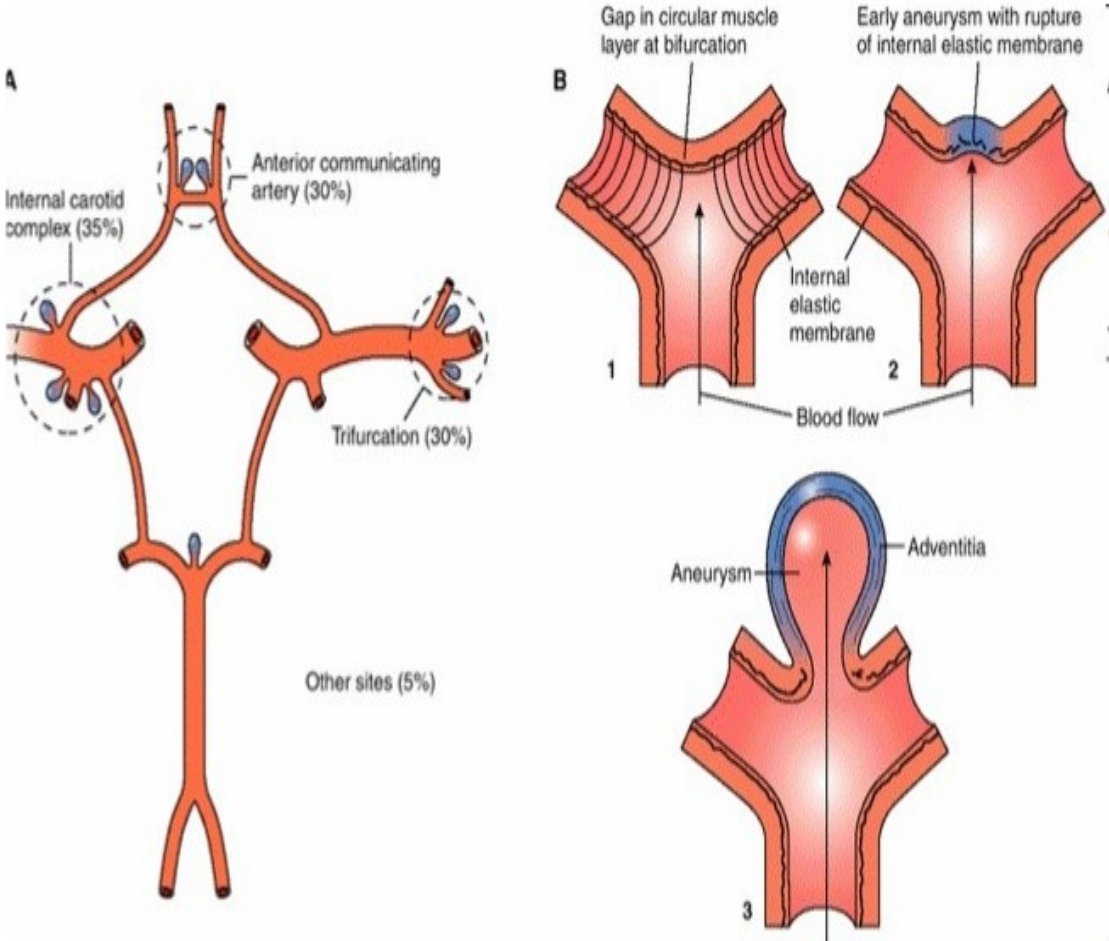
Berry aneurysm on the anterior communicating artery of the brain



ADAM



# Saccular(berry) Aneurysm:



# Cerebral aneurysms



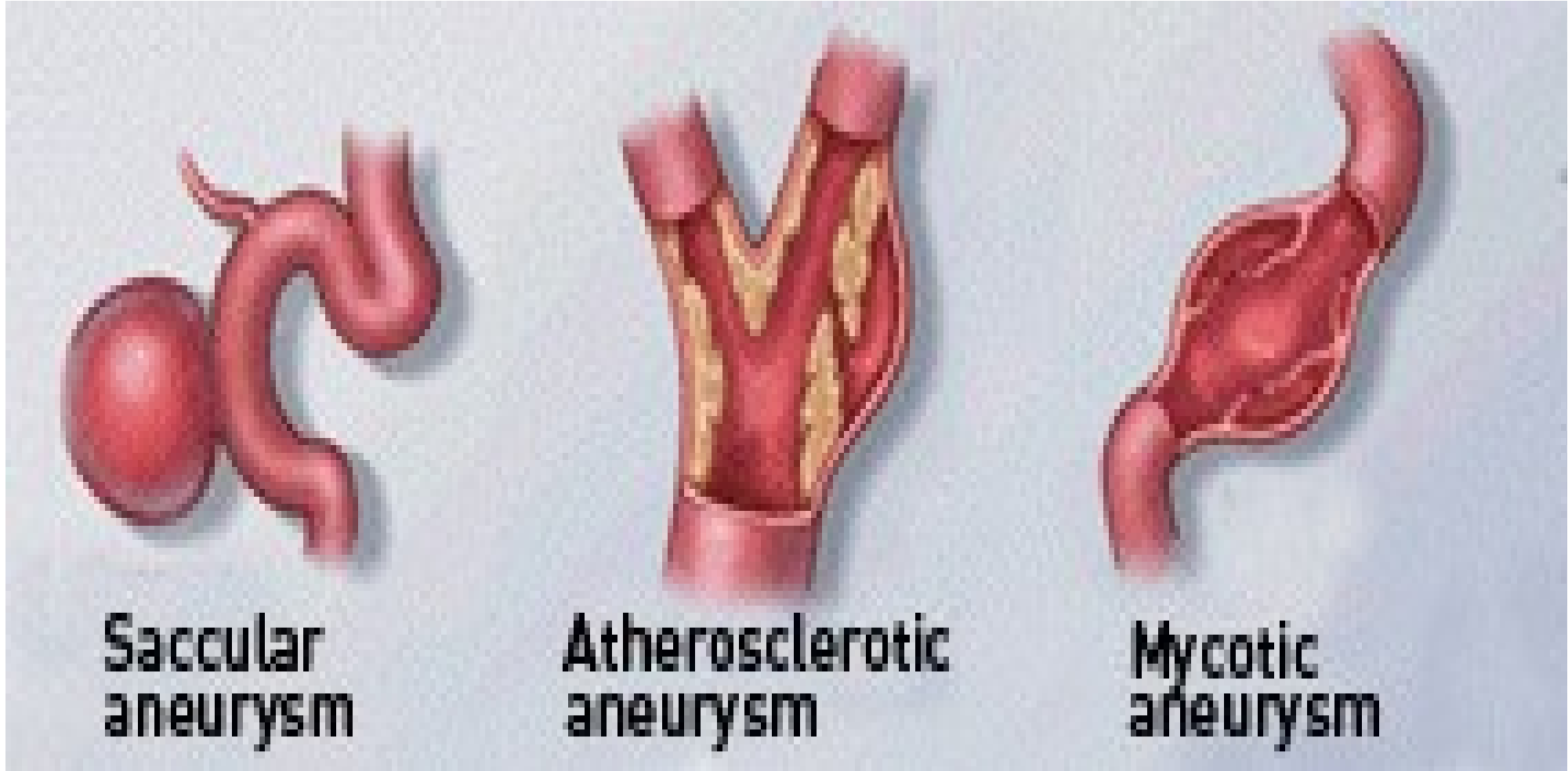
## 2-Mycotic aneurysms:

- Secondary to **inflammatory weakness** in the vascular wall in the circle of Willis.
- They are secondary to complication of **subacute infective endocarditis and polyarteritis nodosa.**

## 3-Atherosclerotic aneurysms:

- Due to **weakening** of media secondary to **atheroma.**
- Common sites are the **basilar and middle cerebral arteries.**

# Cerebral aneurysms



**Saccular  
aneurysm**

**Atherosclerotic  
aneurysm**

**Mycotic  
aneurysm**

<https://tse2.mm.bing.net/th?id=OIP.mc5uPO3EE4njiH-qrKL-eQAAAA&pid=Api&P=0&w=164&h=163>



## Question 1

**The most common cause of Subarachnoid hemorrhage is :**

- a. Traumatic injury of middle meningeal artery.
- b. Rupture of aneurysm of cerebral artery.
- c. Traumatic injury of subdural veins.
- d. Encephalitis.
- e. Anoxia.

## Question 2

Berry aneurysm may be associated with .....



1. Kumar, Vinay, and Abbas, Abul K, and Aster: Robbins Basic Pathology, 10th ed. (2018) Pages 852-856.
2. Mohan H., Mohan P., Mohan T & mohan S. (Eds.). (2015)  
Text book of pathology 7 th edition



THANK YOU!